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**Arterial oxygen tensions in mechanically ventilated patients in the intensive care unit**  
*a descriptive study of hyperoxaemia and associations with mortality*

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recumbent position. Airway (Paw) and esophageal (Pes) pressures and flow were continuously recorded during 2 minutes by a data logger (Biopac 150). Then, end-expiratory and end-inspiratory occlusions were performed for 5 seconds each, then respiratory system was slowly inflated at constant flow from the ventilator. Finally, patient was allowed to breathe out freely to atmosphere by using a three-way stop lock by-passing expiratory valve. AC and airway opening pressure (AOP) were determined according to Chen et al. (1) from the pressure-volume curve. EFL was assessed by the atmospheric method (2). Dynamic elastances of chest wall (Edyn,cw) and lung (Edyn,L) were obtained from least square linear regression method over 10 consecutive breaths. Static elastances (Est,cw and Est,L) were determined by classic formulas and also by taking into account AOP (Est,cw\_aop and Est,L\_aop, respectively). The performance of EcW/EL ratio to predict EFL and AC was assessed by area under receiver operating characteristic (AUCROC) curve.

**RESULTS.** Among 21 included ARDS patients EFL was observed in 7 (33%) and AC in 15 (71%). Median AOP was 7.8 (95%CI 7.6-11.7) cmH<sub>2</sub>O. The AUCROCs for EcW/EL ratios to detect EFL and AC are shown in table 1. Edyn,cw/Edyn,L ratio has the best performance to detect EFL (Edyn,cw/Edyn,L ≤ 0.16 100% sensitivity and 85% specificity). Correction for AOP worsened the performance of Est,cw/Est,L ratio. AC was poorly predicted but prediction was improved with AOP correction. AOP values had an AUCROC of 1 (0.84-1) (P < 0.0001) to predict EFL at the threshold of 8.3 cmH<sub>2</sub>O.

Table 1. Area under ROC curve (95% confidence intervals) for EcW/EL ratio for EFL and AC prediction

**CONCLUSION.** Low Edyn,cw/Edyn,L ratio and AOP predicted EFL occurrence. However, EFL and AC are two distinct phenomena in ARDS patients and should be assessed together to identify specific ARDS subphenotypes in terms of small airway contribution in ARDS.

#### REFERENCE

1. Chen et al AJRCCM 2018 2. Yonis et al AJRCCM 2018

**Table 1 (abstract 000048).** Area under ROC curve (95% confidence intervals) for EcW/EL ratio for EFL and AC prediction

	EFL	P value	AC	P value
Edyn,cw/Edyn,L	0.90 (0.84-0.94)	<0.0001	0.53 (0.45-0.61)	0.51
Est,cw/Est,L	0.79 (0.56-0.93)	0.0099	0.56 (0.33-0.77)	0.69
Est,cw/Est,L_aop	0.57 (0.34-0.78)	0.71	0.79 (0.56-0.94)	0.004

#### 000247

##### Arterial oxygen tensions in mechanically ventilated patients in the intensive care unit: a descriptive study of hyperoxaemia and associations with mortality

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**INTRODUCTION.** Oxygen therapy is part of the invasive mechanical ventilation strategy in the intensive care unit (ICU). Hyperoxaemia may however increase mortality.

**OBJECTIVES.** To assess degrees of hyperoxaemia, adjustments of fraction of inspired oxygen (FiO<sub>2</sub>) in response to hyperoxaemia, and associations between hyperoxaemia and mortality in invasively mechanically ventilated ICU patients.

**METHODS.** We conducted a retrospective study of all invasively mechanically ventilated patients in 5 ICUs admitted from January 2011 to June 2016, retrieving all oxygen tension (PaO<sub>2</sub>) and FiO<sub>2</sub> data. Time between arterial blood gas (ABG) samplings, proportions of hyperoxaemia (PaO<sub>2</sub> > 16 kPa), FiO<sub>2</sub> adjustments upon hyperoxaemia, and associations between mechanically ventilated exposure-time-divided area-under-the-curve (AUC) PaO<sub>2</sub> and ICU mortality was assessed. Primary ICU admissions were included in the mortality analyses, which used a multi-state illness-death model with transition intensities estimated by Cox proportional hazards models. The exposure-time-divided AUC measure represents the average PaO<sub>2</sub> level during invasive mechanical ventilation. Permission to use data without patient consent was obtained from the Danish Patient Safety Authority.

**RESULTS.** The study included 4,998 patients and 177,769 ABGs. Median time between ABGs was 3 hours (inter-quartile range: 2-4 hours), PaO<sub>2</sub> was 11.3 kPa (9.8-13.6 kPa), and FiO<sub>2</sub> was 0.40 (0.35-0.50). Hyperoxaemia was present in 11.9% of the ABGs. In 15.4% of the hyperoxaemic ABGs with FiO<sub>2</sub> ≥ 0.50, the subsequent ABG remained hyperoxaemic with uncorrected FiO<sub>2</sub>, whereas this was the case in 64.6% of hyperoxaemic ABGs with FiO<sub>2</sub> < 0.40. Exposure-time-divided AUC PaO<sub>2</sub> > 16.0 kPa was associated with increased ICU mortality (Table 1). This association was accentuated when censoring at the first PaO<sub>2</sub> < 8 kPa to diminish the impact of hypoxaemia (adjusted HR: 1.90 (95% CI: 1.27-2.86)).

**CONCLUSION.** ABG samplings were frequent and hyperoxaemia was common in invasively mechanically ventilated ICU patients. While FiO<sub>2</sub> in general was adjusted upon hyperoxaemia, several patients remained hyperoxaemic, especially at FiO<sub>2</sub> < 0.40. This may not be optimal as hyperoxaemia was associated with increased ICU mortality in the cohort.

#### REFERENCE

1. Olav L. Schjørring's PhD study was funded by a grant from Innovation Fund Denmark

**Table 1 (abstract 000247).** Hazard ratio (HR) estimates for death in the ICU

Exposure-time-divided AUC PaO <sub>2</sub>	Deaths per 100 person-days (deaths/person-days)	Adjusted HR** (95% CI)
≥ 8.0 kPa to < 12.0 kPa*	2.1 (256/11,930)	1.0
< 8.0 kPa	16.2 (9/55)	6.24 (3.17-12.25)
≥ 12.0 kPa to ≤ 16.0 kPa	2.0 (164/8,010)	1.00 (0.82-1.23)
> 16.0 kPa	3.4 (54/1,571)	1.66 (1.20-2.29)

Among 3,021 patients alive and on ICU 24 hours after primary ICU admission.

\*Reference \*\*Adjusted for gender, age, admission type, Simplified Acute Physiology Score II, being currently ventilated, renal replacement therapy within the first 24 hours of ICU admission, and inotropes or vasopressors within the first 24 hours of ICU admission

#### 000875

##### Positive end-expiratory pressure affects the position and contractile efficiency of the human diaphragm: MRI and functional analysis

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